09/988,292.

WEST

Freeform Search

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Term: L5 and antibod$3

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Search History

DATE: Thursday, January 09, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L6	L5 and antibod$3	3	L6
L5	L4 and human and colon cancer$1	3	L5
L4	galectin 8 or galectin 9 or galectin 10	25	L4
L3	galectin-4-like protein$1	2	L3
L2	L1 and human and antibod$3	4	L2
DB=DWPI,USPT,EPAB,JPAB; PLUR=YES; OP=ADJ			
L1	colon specific gene protein	5	L1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 4 of 4 returned.

☐ 1. 6337195 . 31 Mar 98; 08 Jan 02. Colon specific genes and proteins. Yu; Guo-Liang, et al. 435/70.1; 530/350 536/22.1. C07H021/04 C07K013/00 C12P021/04.

☐ 2. 6080722 . 29 Sep 98; 27 Jun 00. Colon specific gene and protein. Soppet; Daniel R., et al. 514/12; 435/252.3 435/320.1 435/69.1 530/350 536/23.1 536/23.5. A61K038/00 C07K001/00 C12P021/06 C12N001/20 C12N015/00.

☐ 3. 5861494 . 06 Jun 95; 19 Jan 99. Colon specific gene and protein. Soppet; Daniel R., et al. 536/23.1; 435/6 536/24.3. C07H021/02 C07H021/04 C12Q001/68.

☐ 4. 5733748 . 06 Jun 95; 31 Mar 98. Colon specific genes and proteins. Yu; Guo-Liang, et al. 435/70.1; 435/252.3 435/320.1 435/325 536/22.1 536/23.5. C07H021/04.

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Term	Documents
HUMAN.DWPI,EPAB,JPAB,USPT.	439520
HUMANS.DWPI,EPAB,JPAB,USPT.	92952
ANTIBOD$3	0
ANTIBOD.DWPI,EPAB,JPAB,USPT.	331
ANTIBODAY.DWPI,EPAB,JPAB,USPT.	1
ANTIBODEES.DWPI,EPAB,JPAB,USPT.	1
ANTIBODEIS.DWPI,EPAB,JPAB,USPT.	2
ANTIBODES.DWPI,EPAB,JPAB,USPT.	70
ANTIBODES:.DWPI,EPAB,JPAB,USPT.	1
ANTIBODEY.DWPI,EPAB,JPAB,USPT.	1
ANTIBODI.DWPI,EPAB,JPAB,USPT.	8
(L1 AND HUMAN AND ANTIBOD$3).USPT,JPAB,EPAB,DWPI.	4

There are more results than shown above. Click here to view the entire set.

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Search Results - Record(s) 1 through 2 of 2 returned.

☐ 1. EP 841393 A1 . 09 Jul 96. 13 May 98. HUMAN GALECTIN-4-LIKE PROTEIN AND cDNA ENCODING THE SAME. KATO, SEISHI, et al. C12N015/12; C12N015/62 C12N015/63 C12P021/02.

☐ 2. WO 9703190 A1 . 09 Jul 96. 30 Jan 97. HUMAN GALECTIN-4-LIKE PROTEIN AND cDNA ENCODING THE SAME. KATO, SEISHI, et al. C12N015/12; C12N015/62 C12N015/63 C12P021/02.

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Term	Documents
GALECTIN-4-LIKE.DWPI,EPAB,JPAB,USPT.	2
GALECTIN-4-LIKES	0
PROTEIN$1	0
PROTEIN.DWPI,EPAB,JPAB,USPT.	10940
PROTEINE.DWPI,EPAB,JPAB,USPT.	15
PROTEINS.DWPI,EPAB,JPAB,USPT.	6105
PROTEIN/.DWPI,EPAB,JPAB,USPT.	1
PROTEIN:.DWPI,EPAB,JPAB,USPT.	13
(GALECTIN-4-LIKE ADJ PROTEIN$1).USPT,JPAB,EPAB,DWPI.	2
(GALECTIN-4-LIKE PROTEIN$1).USPT,JPAB,EPAB,DWPI.	2

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☐ Generate Collection

L6: Entry 1 of 3 File: USPT Jan 1, 2002

US-PAT-NO: 6335170
DOCUMENT-IDENTIFIER: US 6335170 B1

TITLE: Gene expression in bladder tumors

DATE-ISSUED: January 1, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Orntoft; Torben F.	DK 8230 Aabyhoj			DK

APPL-NO: 09/ 510643 [PALM]
DATE FILED: February 22, 2000

PARENT-CASE:
This application claims the benefit of U.S. Provisional Application No. 60/121,124, filed Feb. 22, 1999, which is hereby incorporated by reference in its entirety.

INT-CL: [07] C12 Q 1/68, C12 P 19/34, C07 H 21/02

US-CL-ISSUED: 435/6; 435/91.1, 435/91.2, 536/23.1, 536/24.3, 536/24.31, 536/24.33
US-CL-CURRENT: 435/6; 435/91.1, 435/91.2, 536/23.1, 536/24.3, 536/24.31, 536/24.33

FIELD-OF-SEARCH: 435/6, 435/91.1, 435/91.2, 536/23.1, 536/24.3, 536/24.31, 536/24.33

PRIOR-ART-DISCLOSED:

U.S. PATENT DOCUMENTS

Search Selected Search ALL

	PAT-NO	ISSUE-DATE	PATENTEE-NAME	US-CL
☐	5677125	October 1997	Holt et al.	435/6
☐	5700637	December 1997	Southern	435/6

FOREIGN PATENT DOCUMENTS

FOREIGN-PAT-NO	PUBN-DATE	COUNTRY	US-CL
198 18 619	October 1999	DE	
89/10977	November 1989	WO	
96/30389	October 1996	WO	
97/16206	May 1997	WO	
97/28446	August 1997	WO	
98/53319	November 1998	WO	
99/47674	September 1999	WO	

OTHER PUBLICATIONS

Liebert et al, "Identification of new biomarkers for bladder cancer using the differential display reverse transcriptase polymerase chain reaction", Proc. Am. Assn. Cancer Res. 38:287, Abstract 1928, Mar. 1997.*
Liebert et al, "Novel molecular markers fof bladder cancer revealed by differential display reverse transcriptase polymerase chain reaction", J. Urol. 159(5 suppl.) 286, Abstract 1101, May 1998.*
Peter S. Nelson, et al., "An Expressed-Sequence-Tag Database of the Human Prostate: Sequence Analysis of 1168 cDNA Clones", Genomics 47, pp. 12-25, 1998.
David B. Krizman, et al., "Construction of a Representative cDNA Library from Prostatic Intraepithelial Neoplasia", Cancer Research 56, pp. 5380-5383, Dec. 1, 1996.

Victoria Hawkins, et al., "PEDB: The Prostate Expression Database", Nucleic Acids Research, vol. 27,No. 1, pp. 240-208, 1999.
Lin Zhang, et al., "Gene Expression Profiles in Normal and Cancer Cells", Science, vol. 276,pp. 1268-1272, May 23, 1997.
Torben F. Orntoft, et al., "Molecular Alterations in Bladder Cancer", United Editorial, XP-000971351, Nov. 11, 1997.
Margaret A. Knowles, et al., Molecular Genetics of Bladder Cancer: Pathways of Development and Progression, Cancer Surveys, vol. 31, pp. 49-76, 1998.

ART-UNIT: 1655

PRIMARY-EXAMINER: Fredman; Jeffrey

ABSTRACT:

Methods for analyzing tumor cells, particularly bladder tumor cells employ gene expression analysis of samples. Gene expression patterns are formed and compared to reference patterns. Alternatively gene expression patterns are manipulated to exclude genes which are expressed in contaminating cell populations. Another alternative employs subtraction of the expression of genes which are expressed in contaminating cell types. These methods provide improved accuracy as well as alternative basis for analysis from diagnostic and prognostic tools currently available.

21 Claims, 24 Drawing figures

End of Result Set

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L6: Entry 3 of 3 File: DWPI Apr 11, 2002

DERWENT-ACC-NO: 2002-507213
DERWENT-WEEK: 200254

TITLE: Diagnosing, monitoring, staging, imaging and treating cancers, e.g. gastrointestinal cancers such as stomach, small intestine and colon cancer, associated with the expression of gastrointestinal specific genes Cln114 and Cln115

INVENTOR: MACINA, R A; PIDERIT, A ; SUN, Y

PATENT-ASSIGNEE: MACINA R A (MACII), PIDERIT A (PIDEI), SUN Y (SUNYI)

PRIORITY-DATA: 2000US-188061P (March 9, 2000), 2001US-0802674 (March 9, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20020042088 A1	April 11, 2002		001	G01N033/574

APPLICATION-DATA:

PUB-NO	APPL-DATE	APPL-NO	DESCRIPTOR
US20020042088A1	March 9, 2000	2000US-188061P	Provisional
US20020042088A1	March 9, 2001	2001US-0802674	

INT-CL (IPC): A61 K 49/16; A61 K 51/10; G01 N 33/574

ABSTRACTED-PUB-NO: US20020042088A
BASIC-ABSTRACT:

NOVELTY - Assays for detecting, diagnosing, monitoring, staging, prognosticating, imaging and treating cancers, particularly gastrointestinal cancers including stomach, small intestine and colon cancer, associated with the expression of gastrointestinal specific genes (GSGs) (specifically Cln114 and Cln115), are new.

DETAILED DESCRIPTION - A method (I) for diagnosing the presence of gastrointestinal cancer in a patient comprising: (a) determining levels of GSG in cells, tissues or bodily fluids in a patient; and (b) comparing the determined levels of GSG with levels of GSG in cells, tissues or bodily fluids from a normal human control (a change in determined levels of GSG in the patient versus normal human control is associated with the presence of gastrointestinal cancer).

INDEPENDENT CLAIMS are also included for the following:

(1) a method of identifying potential therapeutic agents for use in imaging and treating gastrointestinal cancer comprising screening molecules for an ability to bind to GSG (in which the ability of a molecule to bind to GSG is indicative of the molecule being useful in imaging and treating gastrointestinal cancer);

(2) a method (III) of imaging gastrointestinal cancer in a patient comprising administering to the patient an antibody raised against GSG; and

(3) a method (IV) of treating gastrointestinal cancer in a patient comprising administering to the patient an agent which up regulates expression or activity of GSG.

End of Result Set

☐ Generate Collection

L6: Entry 3 of 3 File: DWPI Apr 11, 2002

DERWENT-ACC-NO: 2002-507213
DERWENT-WEEK: 200254

TITLE: Diagnosing, monitoring, staging, imaging and treating cancers, e.g. gastrointestinal cancers such as stomach, small intestine and colon cancer, associated with the expression of gastrointestinal specific genes Cln114 and Cln115

Basic Abstract Text (1):
NOVELTY - Assays for detecting, diagnosing, monitoring, staging, prognosticating, imaging and treating cancers, particularly gastrointestinal cancers including stomach, small intestine and colon cancer, associated with the expression of gastrointestinal specific genes (GSGs) (specifically Cln114 and Cln115), are new.

Basic Abstract Text (2):
DETAILED DESCRIPTION - A method (I) for diagnosing the presence of gastrointestinal cancer in a patient comprising: (a) determining levels of GSG in cells, tissues or bodily fluids in a patient; and (b) comparing the determined levels of GSG with levels of GSG in cells, tissues or bodily fluids from a normal human control (a change in determined levels of GSG in the patient versus normal human control is associated with the presence of gastrointestinal cancer).

Basic Abstract Text (5):
(2) a method (III) of imaging gastrointestinal cancer in a patient comprising administering to the patient an antibody raised against GSG; and

Basic Abstract Text (9):
MECHANISM OF ACTION - Antibody inhibition; vaccine.

Basic Abstract Text (11):
USE - The assays are used for detecting, diagnosing, monitoring, staging, prognosticating, imaging and treating cancers, particularly gastrointestinal cancers including stomach, small intestine and colon cancer, associated with the expression of GSGs, more specifically Cln114 and Cln115 (claimed).

Basic Abstract Text (12):
ADVANTAGE - It has been found that galectin-4 and human carbonic anhydrase I serve as useful markers in the diagnosis of gastrointestinal cancer. These diagnostic markers are referred to as gastrointestinal specific genes (GSGs) and more specifically as Cln114 (galectin-4) and Cln115 (human carbonic anhydrase I). Cln114 was identified as human galectin-4 of 323 amino acids (translated molecular weight of 35918 Dalton). Galectin-4 belongs to the galectin family that include galectin-1 and galectin-3. Both galectin-3 and galectin-4 are found at high concentrations in intestinal extracts. Galectin-4 contains two beta-galactosidase-binding domains and localizes mainly at sites of cell adhesion. Galectin-4 is a cytosolic protein but like galectin-1 and galectin-3 may be externalized by non-classical secretory mechanisms and released from the cell. It is possible that galectin-4 is involved in cell adhesion via interaction with extracellular glyco-conjugates. Galectin-4 has been suggested to play an important role in the maintenance of epithelial integrity and in the epithelial wound healing process (Huflejt et al. J. Biol. Chem. 1997 272(22):14294-303). WO09822139 describes differential expression of galectin-4 in human breast tumor cells. Other members of the galectin superfamily including galectin 8, 9, 10 and 10SV have been described as markers in the diagnosis of cancers including Hodgkin's disease, breast, ovarian, prostate, bone, liver, lung, pancreatic and splenic (see WO 98/15624). In particular GSG refers to native protein expressed by the gene comprising defined

polynucleotide sequences (N1) and (N2) given in the specification. Amino acid sequences encoded by the polynucleotides of N1 and N2 are 2 defined 323 and 261 amino acid sequences (A1) and (A2) given in the specification (respectively).

Standard Title Terms (1):
DIAGNOSE MONITOR STAGE IMAGE TREAT CANCER GASTRO CANCER STOMACH INTESTINAL COLON CANCER ASSOCIATE EXPRESS GASTRO SPECIFIC GENE

ACTIVITY - Cytostatic.

No biological data given.

MECHANISM OF ACTION - Antibody inhibition; vaccine.

No supporting data provided.

USE - The assays are used for detecting, diagnosing, monitoring, staging, prognosticating, imaging and treating cancers, particularly gastrointestinal cancers including stomach, small intestine and colon cancer, associated with the expression of GSGs, more specifically Cln114 and Cln115 (claimed).

ADVANTAGE - It has been found that galectin-4 and human carbonic anhydrase I serve as useful markers in the diagnosis of gastrointestinal cancer. These diagnostic markers are referred to as gastrointestinal specific genes (GSGs) and more specifically as Cln114 (galectin-4) and Cln115 (human carbonic anhydrase I). Cln114 was identified as human galectin-4 of 323 amino acids (translated molecular weight of 35918 Dalton). Galectin-4 belongs to the galectin family that include galectin-1 and galectin-3. Both galectin-3 and galectin-4 are found at high concentrations in intestinal extracts. Galectin-4 contains two beta-galactosidase-binding domains and localizes mainly at sites of cell adhesion. Galectin-4 is a cytosolic protein but like galectin-1 and galectin-3 may be externalized by non-classical secretory mechanisms and released from the cell. It is possible that galectin-4 is involved in cell adhesion via interaction with extracellular glyco-conjugates. Galectin-4 has been suggested to play an important role in the maintenance of epithelial integrity and in the epithelial wound healing process (Huflejt et al. J. Biol. Chem. 1997 272(22):14294-303). WO09822139 describes differential expression of galectin-4 in human breast tumor cells. Other members of the galectin superfamily including galectin 8, 9, 10 and 10SV have been described as markers in the diagnosis of cancers including Hodgkin's disease, breast, ovarian, prostate, bone, liver, lung, pancreatic and splenic (see WO 98/15624). In particular GSG refers to native protein expressed by the gene comprising defined polynucleotide sequences (N1) and (N2) given in the specification. Amino acid sequences encoded by the polynucleotides of N1 and N2 are 2 defined 323 and 261 amino acid sequences (A1) and (A2) given in the specification (respectively).

ABSTRACTED-PUB-NO: US20020042088A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/0

DERWENT-CLASS: B04 D16 S03
CPI-CODES: B04-E03F; B04-G01; B04-N02; B11-C07A3; B12-K04A1; B12-K04E; D05-H09; D05-H11; D05-H12A; D05-H17A;
EPI-CODES: S03-E14H4;

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L6: Entry 2 of 3 File: USPT Jul 3, 2001

US-PAT-NO: 6255049
DOCUMENT-IDENTIFIER: US 6255049 B1

TITLE: Detection of metastatic cancer cells using PCTA-1

DATE-ISSUED: July 3, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Fisher; Paul B.	Scarsdale	NY		

ASSIGNEE-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY	TYPE CODE
The Trustees of Columbia University in the City of New York	New York	NY			02

APPL-NO: 09/ 032311 [PALM]
DATE FILED: February 27, 1998

INT-CL: [07] C12 Q 1/68, C12 P 19/34, C07 H 21/04

US-CL-ISSUED: 435/6; 435/91.2, 435/91.5, 435/91.51, 536/23.5, 536/24.31, 536/24.33
US-CL-CURRENT: 435/6; 435/91.2, 435/91.5, 435/91.51, 536/23.5, 536/24.31, 536/24.33

FIELD-OF-SEARCH: 435/6, 435/91.1, 435/91.2, 435/91.5, 435/91.51, 536/24.3, 536/24.31, 536/24.33, 536/23.5

PRIOR-ART-DISCLOSED:

FOREIGN PATENT DOCUMENTS

FOREIGN-PAT-NO	PUBN-DATE	COUNTRY	US-CL
96 21671	July 1996	WO	

OTHER PUBLICATIONS

Fisher, P., B., (1995) "A new technology for preparig monoclonal antibodies to molecules expressed on the cell surface", Pharmaceutical Tech. 19(9):42-48, (Exhibit 2).
Gronberg, H., et al., (1997) "Early age at diagnosis in families provided evidence of linkage to the hereditary prostate cancer locus (HPCI) on chromosome 1", Cancer Res. 57:4707-4709, (Exhibit 3).
Raz, A. and Lotan, R., (1987) "Endogenous galactoside-binding lectins: a new class of functional tumor or cell surface molecules related to metastasis", Cancer Metastasis Rev, 6:433-452, (Exhibit 4)
Shen, R., et al., (1994) "Surface epitope masking: a strategy for the development of monoclonal antibodies specific for molecules expressed on the cell surface", J. Natl. Cancer Inst. 86:91-98, (Exhibit 5).
Smith, J.R., et al., (1996) "Major susceptibility locus for prostate cancer on chromosome 1 suggested by a genome-wide search", Science 274:1371-1375, (Exhibit 6).
Su, Z-Z, et al., (1996) "Surface-epitope masking and expression cloning identifies the human prostate carcinoma tumor antigen gene PCTA-1 a member of the galectin gene family", Proc. Natl. Acad. Sci. U.S.A. 93:7252-7257, (Exhibit 7).

Su, Zao-Zhong, Goldstein, Neil I. and Fisher, Paul B., (1998) "Antisense inhibition of the PTI-1 oncogene reverses cancer phenotypes", Proc. Natl. Acad. Sci. USA 95:1-6, (Exhibit 8); and.
Sun, Y., et al., (1997) "Human prostatic carcinoma oncogene PTI-1 is expressed in human tumor cell lines and prostate carcinoma patient blood samples", Cancer Res. 57:18-23, (Exhibit 9).
Sokoloff, M.H. et al. Quantitative PCR does not improve preoperative prostate cancer staging: a clinicopathological molecular analysis of 121 patients. J. Urology 156:1560-1566, 1996.*
Heid, C.A. et al. Real time quantitative PCR. Genome Research 6:986-994, 1996.*
Hadari, Y.R. et al. Galectin-8. J. Biol. Chem. 270(7):3447-3453, 1995.*
Hadari, Y.R. Galectin-8: on the road from structure to function. Trends Glycos. Glycotech. 9(45):103-112, Jan. 1997.*
Caulet-Maugendre, S. et al. Galectin-8 expression in squamous cell metaplasia of the bronchial epithelium in squamous cell cancer and benign processes. Modern Pathology 11(1):A172, Jan. 1998.*
Sokoloff, M. et al. Super-sensitive and quantitative PCR: an innovative technique for staging and monitoring prostate cancer. J. Urol. 153(4) Suppl:294A, 1995.

ART-UNIT: 165

PRIMARY-EXAMINER: Myers; Carla J.

ASSISTANT-EXAMINER: Johanssen; Diana

ABSTRACT:

This invention provides a method of detecting cancer metastatic cells in a subject, comprising: a) obtaining a nucleic acid sample from the subject's blood; b) amplifying nucleic acid encoding the product of prostate carcinoma tumor antigen gene-1; and c) detecting the presence of nucleic acid encoding the product of the prostate carcinoma tumor antigen gene-1, thereby detecting cancer metastatic cells in a subject. This invention also provides the above-described methods, wherein the method of amplification is PCR. This invention further provides the above-described methods, wherein the primers are 5'-AAGCTGACGCCTCATTTGCA-3' SEQ ID NO: 1 and 5'-AACCACCAATGGAACTGGGT-3' SEQ ID NO: 2. This invention also provides the above-described methods, wherein the primers are 5'-AATGGCTTCTGTGATACT-3' SEQ ID NO: 3 and 5'-GGCTATAAGTGTTGCTGC-3'SEQ ID NO: 4.

4 Claims, 9 Drawing figures

☐ Generate Collection

L6: Entry 2 of 3 File: USPT Jul 3, 2001

DOCUMENT-IDENTIFIER: US 6255049 B1
TITLE: Detection of metastatic cancer cells using PCTA-1

Government Interest Text (1):
The invention disclosed herein was made with Government support under National Cancer Institute Grant Nos. CA35675 and CA21124 from the Department of Health and Human Services Accordingly, the U.S. Government has certain rights in this invention.

Brief Summary Text (4):
Two novel approaches have been developed for defining the molecular and immunological basis of human cancer. One approach, the REXCS (2,3), permits the identification of putative oncogenic elements in human cancer DNAs. RExCS involves cotransfection of high molecular weight human tumor DNA in combination with a selectable antibiotic resistance gene into a new DNA acceptor cell line, CREF-Trans 6, selecting antibiotic resistant cells, injecting these cells into nude mice, and establishing cell cultures from tumors developing in athymic nude mice (2,3). Tumor-derived cell lines are then used to identify the putative human oncogenes, using approaches such as differential RNA display, that have been transferred from the human cancer cells into CREF-Trans 6 (3). Another methodology, SEM (7,8), results in the efficient and selective production of monoclonal antibodies reacting with tumor associated antigens present on the surface of human cancer cells (4,7,8). This procedure involves immunological subtraction based on the selective blocking of surface antigens on genetically altered cells (referred to as a "tester") with high titer polyclonal antibodies produced against untransfected parental cells (referred to as a "driver") This is followed by the production of hybridomas secreting MAbs that react with defined as well as uncharacterized cell surface expressed molecules (7,8).

Brief Summary Text (5):
Using SEM in combination with RExCS, prostate carcinoma (Pro 1.5) MAbs have been developed that react with TAAs expressed on human prostate cancer cell lines and patient-derived carcinomas (4,7,8). Addition of the Pro 1.5 MAb to nude mice containing human prostate cancer xenografts suppressed tumor growth and cancer progression. Screening a human LNCaP prostate cancer cDNA expression library with the Pro 1.5 MAb identified PCTA-1, a gene that encodes a novel galactose binding lectin, designated galectin-8HT (7). PCTA-1 was previously described in International Application No. PCT/US96/00307, filed Jan. 11, 1996, and published on Jul. 18, 1996 under International Publication No. WO 96/21671, which is herein incorporated by reference.

Brief Summary Text (6):
Using primer pairs corresponding to the 3' untranslated (UTR) region, RT-PCR detected PCTA-1 expression in prostatic carcinomas and PIN, but not in histologically confirmed normal prostate or BPH (7). Although further studies are necessary using a larger number of patient samples, these provocative results suggest that the Pro series of murine MAbs and the PCTA-1 gene should have direct diagnostic applications for human prostate cancer. Moreover, if the Pro MAbs show specificity toward prostate cancer, appropriately modified Pro MAbs, including chimerized and humanized versions of this MAb, may have therapeutic potential in patients with primary and metastatic prostate cancer.

Drawing Description Text (12):
FIG. 4A Presence of the PCTA-1 gene in a panel of 19 rodent-human hybrids. A fully shaded box indicates that the hybrid named in the left column contains the chromosome indicated in the upper row; a box shaded in the lower right indicates the presence of the long arm of the chromosome (or part of the long arm represented by a smaller fraction of stippling); a box shaded in the upper left indicates the presence of the

short arm (or partial short arm) of the chromosome; an unshaded box indicates the absence of the chromosome listed above the column. The column for chromosome 1 is outlined in bold and stippled to highlight correlation of presence of the PCTA-1 gene. The pattern of retention of the PCTA-1 locus in the panel is shown to the right of the figure where presence of the locus in a hybrid is indicated by a stippled box with a plus sign and absence of the locus is indicated by an open box enclosing a minus sign.

Detailed Description Text (6):
This invention provides a method of detecting colon cancer metastasis in a subject, comprising: a) obtaining a nucleic acid sample from the subject's blood; b) amplifying nucleic acid encoding the product of prostate carcinoma tumor antigen gene-1; and c) detecting the presence of nucleic acid encoding the product of the prostate carcinoma tumor antigen gene-1, thereby detecting colon cancer metastasis in a subject.

Detailed Description Text (13):
This invention also provides a method of determining the stage of colon cancer in a subject, comprising: a) obtaining a nucleic acid sample from the subject's blood; b) amplifying the nucleic acid encoding the product of prostate carcinoma tumor antigen gene-1; and c) quantifying the levels of RNA encoding the product of prostate carcinoma tumor antigen gene-1, thereby determining the stage of colon cancer in a subject.

Detailed Description Text (34):
Probes that produce signals that amplify detection of the probe include: radioactively labeled probes, enzyme-linked probes, and certain forms of antibody-linked and/or antigen-linked probes. Radioactive and enzyme-linked probes produce increasing amounts of signal over time, thus amplifying detection of the probe. Antigen-linked probes allow for antibodies to react with the probe, thus, amplifying the detection of the probe. Similarly for antibody-linked probes.

Detailed Description Text (44):
This invention also provides the above-described methods, wherein the cancer is colon cancer.

Detailed Description Text (49):
The abbreviations used herein are: SEM, surface-epitope masking; PCTA-1, prostate carcinoma tumor antigen gene-1; BPH, benign prostatic hypertrophy; PIN, prostatic intraepithelial neoplasia; TAAs, tumor associated antigens; RExCS, rapid expression cloning system; MAbs, monoclonal antibodies; 3' UTR, 3' untranslated region; RER, replication error phenotype; LOH, loss of heterozygosity.

Detailed Description Text (50):
An immunological subtraction approach, named surface-epitope masking (SEM), and antibody expression cloning identified a novel gene, prostate carcinoma tumor antigen gene-1 (PCTA-1), with restricted expression in human prostate cancer (Su et al., PNAS, 93: 7252-7257, 1996). Using Northern blotting, RNase protection and RT-PCR we document the specificity of PCTA-1 expression to neoplastic prostate. We also demonstrate that PCTA-1 can be used as a sensitive monitor to detect potentially metastatic cancer cells in the circulation of patients with confirmed metastatic prostate disease. These findings suggest that PCTA-1 may prove useful as a sensitive diagnostic marker for determining prostate cancer progression as indicated by the escape of prostate carcinoma cells from the prostate gland into the bloodstream. PCTA-1 is encoded by a gene located at 1q42-43, an area previously identified as a replication error-type genetic instability locus in human male germ cell tumors. In these contexts, PCTA-1 may play a role in normal germinal cell development and abnormalities in PCTA-1 expression may contribute to the etiology and progression of human prostate cancer.

Detailed Description Text (51):
In the present study we provide definitive evidence for the specificity of PCTA-1 expression in neoplastic prostate. PCTA-1 RNA is detected using Northern blotting, RNase protection and RT-PCR assays in prostate carcinoma cell lines and tissues from patients with cancer, but not in normal prostate or BPH tissues. Data is also presented indicating that PCTA-1 can be detected using RT-PCR when one PCTA-1 expressing cell is diluted in the equivalent of ten million non-PCTA-1 expressing

cells. In this context, RT-PCR using PCTA-1 can detect putative metastatic prostate carcinoma cells in the blood of patients with metastatic disease. Chromosome mapping indicates that PCTA-1 is located at 1q42-43, a locus previously reported to be genetically unstable in human male germ cell tumors (14). These findings suggest the intriguing possibility that PCTA-1 may contribute to normal germ cell development and abnormalities in its expression may result in prostate and possibly human germ cell neoplasias.

Detailed Description Text (52):
PCTA-1 expression is prostate carcinoma specific. Previous studies using RT-PCR analysis of PCTA-1 expression in prostate carcinoma cell lines and a small panel of putative normal prostate (4 samples), BPH (4 samples) and prostate carcinoma (7 samples) patient-derived tissue suggest a restricted pattern of PCTA-1 expression to neoplastic prostate (4). In this limited analysis, one putative normal prostate sample and one BPH sample were positive for PCTA-1 expression. However, because the putative normal prostate tissue was obtained from a 60-year old-man, without histological evaluation, it is possible that this tissue may have contained clinically unsuspected prostate disease (4). In the one BPH sample positive for PCTA-1 expression, histological analysis indicated the presence of epithelial atypia consistent with high-grade PIN (4). Although suggestive, these studies do not definitively demonstrate a direct association between PCTA-1 expression and human prostate cancer.

Detailed Description Text (53):
In the present study, three molecular approaches, Northern blotting, RNase protection and RT-PCR, have been used to examine PCTA-1 expression in prostate cell lines and tissue samples from normal prostate, BPH, PIN and carcinoma. Northern blotting indicates the presence of two RNA species (.about.3.7 and 1.5 kb) in LNCaP-DNA transfected CREF-Trans 6:4 NMT cells and DU-145, LNCaP and PC-3 human prostate carcinoma cell lines (FIG. 1A). In contrast, PCTA-1 expression is not detected in CREF-Trans 6 or NHPE cells. Analysis of tissue RNA demonstrates that PCTA-1 expression is detected, although at low levels (possibly reflecting the heterogeneous nature of the tissue samples), in four of four prostate carcinoma tissues (FIG. 1A). As observed with cultured NHPE cells, no PCTA-1 expression is apparent in normal prostate (2 samples) or in a BPH (1 sample) tissue sample. Similarly, RNase protection assays detect a protected transcript of the appropriate size (411 nt) in CREF-Trans 6:4 NMT, DU-145, LNCaP and PC-3, but not in CREF-Trans 6 or NHPE (FIG. 1B). The level of expression of PCTA-1 is lowest in PC-3, as previously observed using immunofluorescence and immunoprecipitation analyses (4). Examination of four prostate carcinomas, a normal prostate, a low-grade PIN and two BPH tissue samples by RNase protection assays indicate restricted expression of PCTA-1 to neoplastic prostate tissue (FIG. 1C). Although PCTA-1 can be detected in cancer patient tissue samples by Northern blotting and RNase protection assays, these approaches are not ideal for routine screening of clinical materials. The reasons for this include a limited sensitivity of PCTA-1 detection of prostate cancer cells using these approaches (most likely reflecting the small numbers of cancer cells present in the clinical samples analyzed) and the necessity of using relatively large amounts of RNA, 15 to 20 and 5 .mu.g per assay respectively. This later problem can be particularly prohibitive, especially when only small quantities of clinical material are available. Although the sample size of this study is small, these analyses conclusively demonstrate that PCTA-1 is expressed specifically in prostate carcinoma cell lines and patient-tissues as opposed to normal prostate, BPH or low-grade PIN tissues.

Detailed Description Text (55):
PCTA-1 expression is detected in the blood of patients with metastatic prostate cancer. To define potential applications for using RT-PCR of PCTA-1 in staging and monitoring prostate cancer progression, PCTA-1 expressing CREF-Trans 6:4 NMT (DU-145 or LNCaP) cells were serially diluted with non-PCTA-1 expressing CREF-Trans 6 cells; total RNA was isolated, and samples were compared by RT-PCR (FIG. 2B and data not shown). Using primers designed in the 3' UTR of PCTA-1, a positive PCTA-1-specific amplified fragment (411 nt) is present when 1 CREF-Trans 6:4 NMT (DU-145 or LNCaP) cell is diluted in 10.sup.7 CREF-Trans 6 cells (FIG. 2B and data not shown). This level of sensitivity exceeds that obtained when performing similar RT-PCR assays with LNCaP cells diluted in CREF-Trans 6 cells and monitoring PSA (sensitivity of 1 in 10.sup.6) or prostate-specific membrane antigen (sensitivity of 1 in 10.sup.5) (6). However, the sensitivity of PCTA-1 is 10-fold less than obtained when performing

RT-PCR with the same samples and monitoring for expression of a novel oncogene overexpressed in prostate carcinoma cells, prostate tumor inducing gene-1 (PTI-1) (sensitivity of 1 in 10.sup.8) (3,6,19). These results demonstrate that PCTA-1 is a sensitive detector of human prostate carcinoma cells, exceeding the sensitivity of PSA and PSM.

Detailed Description Text (57):
The human PCTA-1 gene maps to chromosome 1q42-43. Important information relative to the potential function and/or relationship between a specific cDNA and a disease state can be obtained by defining chromosomal localization of the gene. To address this question, DNA from a panel of rodent-human hybrids, together carrying most human chromosome regions, were tested for presence of the PCTA-1 locus by PCR amplification. Hybrid DNAs were scored positive if they exhibited a 177 bp PCTA-1 human specific product. Hybrids retaining chromosome region 1q in common had the PCTA-1 specific product; those without 1q did not have a human PCTA-1 product, as summarized in FIG. 4A and illustrated in FIG. 4B. To further refine the localization of PCTA-1, the Stanford RH panel (Research Genetics) was tested by PCR-amplification for the PCTA-1 fragment. The scoring data was submitted to the Stanford rhserver (rhserver@shgc.stanford.edu) and results calculated by the server showed linkage to two markers (with LOD score greater than 6). D1S235 and D1S446 were linked to PCTA-1 at distances of 16.98 cR-8000 and 33.23 cR-8000, respectively. This data was confirmed after testing the Genebridge radiation hybrid DNAs (Research Genetics). The WICGR mapping server (www.genome.wi.mit.edu/cgi-bin/contig/rhmapper.pl) found very close linkage to WI-9317 (DIS2421) and WI-6955 (DIS2383). Thus, PCTA-1 maps to the chromosome region 1q42-43, where these linked markers have been localized (20).

Detailed Description Text (60):
A number of chromosomal abnormalities are present in cells from human prostate cancer (21-25). These include, duplication of sequences located on the distal arm of chromosome 8 (21) and the loss of DNA sequences from the short arm of chromosomes 8 and 11 and the long arm of chromosome 13 (22,23). Recent studies using linkage analysis with prostate cancer-prone families provides evidence for a region of the long arm of chromosome 1 (1q24-25) as the location of a gene(s) representing a potential site for predisposition to prostate cancer (24,25). This study suggests that chromosome 1, specifically 1q, previously considered not to be an important region in prostate cancer, may in fact be a major susceptibility locus for this disease. In this context, the localization of PCTA-1 to chromosome 1q, specifically 1q42-43, suggests that chromosome 1 may also contain a gene that directly contributes to the cancerous potential of prostate cancer cells.

Detailed Description Text (61):
The RER phenotype, recognized as microsatellite sequence alterations, has been suggested to be associated with cancer development, specifically hereditary nonpolyposis colorectal cancer and several types of sporadic tumors (26). Microsatellite instability reflects a pathway in the development of tumors that can be distinguished from a direct loss of tumor suppressor gene function that is distinctive of many tumor suppressor genes. A high frequency of (CA)n sequence instability is evident in several types of sporadic human tumors, including colorectal (12-28%) (27), endometrial (17%) (28), pancreatic (67%) (29), gastric (22.7-39%) (24), prostate (37.5%) (30), breast (10.6%) (31) and germ cell tumor (18.2%) (14). In the case of germ cell tumors, an .about.18.2% RER type genetic instability occurs at 1q42-43 (14), the location of PCTA-1. It is possible that the genetic instability at 1q42-43 modifies PCTA-1 expression and may mediate or correlate with the development of germ cell tumors. This hypothesis is currently being tested experimentally.

Detailed Description Text (62):
The effective uses of MAbs as diagnostic and therapeutic reagents in cancer require that they embody the appropriate selective specificity toward tumor cells. Surface molecules represent potentially useful targets for MAb-based therapeutics (32-34). Production of MAbs reacting with antigens present on the surface of tumor cells, but displaying restricted expression on normal cells, is often a difficult and inefficient process. The SEM procedure (7,8) has been developed that in principal can obviate many of the limitations preventing efficient MAb development toward molecules expressed on the cell surface. The SEM approach has been successfully used for a number of applications resulting in the production of MAbs specific for surface expressed

molecules with known and unknown functions (4,7,8). We have successfully used SEM in combination with RExCS to develop MAbs reacting with antigens expressed preferentially on the surface of human prostate and breast cancer (4,7,8 and unpublished data). Moreover, repeated injection of the SEM-derived Pro 1.5 MAb into athymic nude mice containing established DU-145 human prostate tumor xenografts inhibits tumor growth and even results in complete tumor remission in specific animals. These provocative findings demonstrate a direct effect of Pro 1.5 MAbs on the growth and progression of human prostate cancers in vivo. The SEM procedure should prove extremely useful in producing MAbs and identifying genes associated with important cellular processes, including growth control, differentiation, senescence, immunologic recognition, tumorigenesis, metastasis, atypical multidrug resistance and autoimmune diseases.

Detailed Description Text (63):
In summary, SEM in combination with RExCS is an extremely sensitive and efficient technology to develop MAbs and for cloning genes expressed differentially in cancer versus normal tissue. The identification of PCTA-1 as a gene associated with human prostate cancer development and evolution provides proof of principle for these applications in identifying a potentially relevant gene in human prostate cancer. The demonstration that PCTA-1 is located on the long arm of chromosome 1 also indicates that this chromosomal region may contain genes responsible for both the susceptibility to prostate cancer and a gene that determines the aggressiveness of prostate cancers.

Detailed Description Text (65):
Cell Lines, Tumor Tissue and Blood Samples. The following cell types were used: early passage normal human prostate epithelial cells (NHPE, passage number <12), prostate carcinoma cells (DU-145, LNCaP and PC-3), cloned rat embryo fibroblast cells (CREF-Trans 6) and nude mouse tumor-derived CREF-Trans 6 cells transfected with LNCaP DNA (CREF-Trans 6:4 NMT). NHPE cells were grown in defined serum-free medium provided by Clonetics (San Diego, Calif.). The different cell lines were grown in DMEM supplemented with 5% (rodent cells) or 10% (human cells) fetal bovine serum at 37.degree. C. in a 95% air/5% CO.sub.2 -humidified incubator. Tissue samples from normal prostates, patients with BPH, low-grade PIN, high-grade PIN and carcinoma were obtained during surgery or from autopsies performed at Columbia Presbyterian Medical Center (Dr. Carl A. Olsson, Department of Urology, Columbia University College of Physicians and Surgeons, New York, N.Y.) and from the Cooperative Human Tumor Network (CHTN). Blood RNAs used in this study were obtained from patients at Columbia-Presbyterian Medical Center and from Dr. Gerald Murphy (Pacific Northwest Cancer Foundation, Seattle, Wash.). Tissue and blood samples were obtained with informed consent of each patient using protocols approved by the Institutional Review Boards. Sample analysis included blood samples from patients with D stage disease, a patient with C stage disease and normal males and a female.

Detailed Description Text (66):
RNA Isolation from Human Prostate Cells, Tissues and Blood Samples. Total RNA was isolated from human normal prostate, BPH, PIN and carcinoma tissue using the TRizol reagent (Life Technologies) as described previously (3). Briefly, tissue samples were frozen in liquid nitrogen and homogenized into powder (3,4). To each gram of tissue, 10 ml of TRizol was added and mixed thoroughly followed by incubation at room temperature for 5 min. To each ml of TRizol added, 0.2 ml of chloroform was added and the reactants were thoroughly mixed. The tubes were centrifuged at 12,000.times.g for 15 min and the aqueous phase was transferred to a fresh tube. RNA was precipitated with iso-propyl alcohol and washed with 75% alcohol. After drying, RNA was dissolved in RNase free water.

Detailed Description Text (69):
Chromosomal Localization. Rodent-human hybrids. Many of the hybrids used in this study are available through the Human Genetic Mutant Repository (HGMCR Coriell Institute, Camden, N.J.) or were described previously (17). Stanford and Genebridge radiation hybrid (RH) mapping panels (18,19) were purchased from Research Genetics (Huntsville, Ala.). PCR amplification. The oligonucleotides for generating PCR products were selected using the computer program Oligo 4.0 (National Biosciences). Primers used to amplify a 177 bp PCTA-1 product were: PCTAF, 5'-AATGGCTTCTGTGATACT-3' SEQ ID NO:3 and PCTAR, 5'-GGCTATAAGTGTTGCTGC-3' SEQ ID NO:4. PCR reactions were carried out in a final volume of 12.5 .mu.l with 100 ng of template, 20 ng primers, 10 mM tris-HCl pH 8.3, 50 mM KCl, 0.1 mg/ml gelatin, 15 mM MgCl2, 200 .mu.M NTPs and 0.5 U Taq polymerase.

Amplifications were performed in a Perkin-Elmer Cetus 9600 thermal cycler for 30 cycles of 94.degree. C. for 30s, 58.degree. C. for 30s and 72.degree. C. for 30s. The PCR products were visualized in ethidium bromide stained 1.5% agarose gels. The amplification product of the gene was sequenced to ensure its identity. The product was purified with Qiagen PCR purification kit, and 1 ng of DNA and 20 ng specific primer used with the Taq Dye Deoxy Terminator Cycle Sequencing Kit (ABI). The reaction products were electrophoresed and recorded on the 377 DNA sequencer (ABI).

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Detailed Description Text (73):
3. Shen, R., Su, Z.-z., Olsson, C. A., and Fisher, P. B. Identification of the human prostatic carcinoma oncogene PTI-1 by rapid expression cloning and differential RNA display. Proc. Natl. Acad. Sci. U.S.A., 92: 6778-6782, 1995.

Detailed Description Text (74):
4. Su, Z.-z., Lin, J., Shen, R., Fisher, P. E., Goldstein, N. I., and Fisher, P. B. Surface-epitope masking and expression cloning identifies the human prostate carcinoma tumor antigen gene PCTA-1 a member of the galectin gene family. Proc. Natl. Acad. Sci. U.S.A., 93: 7252-7257, 1996.

Detailed Description Text (75):
5. Wang, F-L., Wang, Y., Wong, W-K., Liu, Y., Addivinola, J., Liang, P., Chen, L. B., Kantoff, P. W., and Pardee, A. B. Two differentially expressed genes in normal human prostate tissue and in carcinoma. Cancer Res., 56: 3634-3637, 1996.

Detailed Description Text (76):
6. Sun, Y., Lin, J., Katz, A. E., and Fisher, P. B. Human prostatic carcinoma oncogene PTI-1 is expressed in human tumor cell lines and prostate carcinoma patient blood samples. Cancer Res., 57: 18-23, 1997.

Detailed Description Text (77):
7. Shen, R., Su, Z.-z., Olsson, C. A., Goldstein, N. I., and Fisher, P. B. Surface-epitope masking: a strategy for the development of monoclonal antibodies specific for molecules expressed on the cell surface. J. Natl. Cancer Inst., 86: 91-98, 1994.

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33. Waldman, T. A. Monoclonal antibodies in diagnosis and therapy. Science (Washington D.C.), 252: 1657-1662, 1991.

Detailed Description Text (104):
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Other Reference Publication (1):
Fisher, P., B., (1995) "A new technology for preparig monoclonal antibodies to molecules expressed on the cell surface", Pharmaceutical Tech. 19(9):42-48, (Exhibit 2).

Other Reference Publication (4):
Shen, R., et al., (1994) "Surface epitope masking: a strategy for the development of monoclonal antibodies specific for molecules expressed on the cell surface", J. Natl. Cancer Inst. 86:91-98, (Exhibit 5).

Other Reference Publication (6):
Su, Z-Z, et al., (1996) "Surface-epitope masking and expression cloning identifies the human prostate carcinoma tumor antigen gene PCTA-1 a member of the galectin gene family", Proc. Natl. Acad. Sci. U.S.A. 93:7252-7257, (Exhibit 7).

Other Reference Publication (8):
Sun, Y., et al., (1997) "Human prostatic carcinoma oncogene PTI-1 is expressed in human tumor cell lines and prostate carcinoma patient blood samples", Cancer Res. 57:18-23, (Exhibit 9).

Other Reference Publication (11):
Hadari, Y.R. et al. Galectin-8. J. Biol. Chem. 270(7):3447-3453, 1995.*

Other Reference Publication (13):
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CLAIMS:

1. A method of detecting metastatic prostate cancer cells in a human subject's blood which comprises detecting in RNA obtained from cells present in the subject's blood, RNA which encodes prostate carcinoma tumor antigen-1, wherein the presence of said RNA which encodes prostate carcinoma tumor antigen-1 is indicative of the presence of metastatic prostate cancer cells in the human subject's blood.

2. A method of detecting metastatic prostate cancer cells in a human subject's blood, comprising:

a. obtaining RNA from cells present in the subject's blood;

b. admixing the RNA obtained in step (a) with two nucleic acid primers consisting of the sequence 5'-AAGCTGACGCCTCATTTGCA-3' (Seq. ID 1) and 5'-AACCACCAATGGAACTGGGT-3' (Seq. ID 2) and subjecting the mixture to a polymerase chain reaction to produce a

polymerase chain reaction amplified product;

c. contacting the amplified product from step (b) with a labeled probe which specifically hybridizes with nucleic acid encoding prostate carcinoma tumor antigen-1wherein specifically hybridized probe produces a signal; and

d. detecting the presence of said signal, as indicative of the presence of nucleic acid encoding prostate carcinoma tumor antigen-1, wherein the presence of said nucleic acid encoding prostate carcinoma tumor antigen-1 is indicative of the presence of metastatic prostate cancer cells in the human subject's blood.

3. A method of detecting metastatic prostate cancer cells in a human subject's blood, comprising:

a. obtaining RNA from cells present in the subject's blood;

b. admixing the RNA obtained in step (a) with two nucleic acid primers consisting of the sequence 5'-AATGGCTTCTGTGATACT-3' (Seq. ID 3) and 5'-GGCTATAAGTGTTGCTGC-3' (Seq ID 4) and subjecting the mixture to a polymerase chain reaction to produce a polymerase chain reaction amplified product;

c. contacting the amplified product from step (b) with a labeled probe which specifically hybridizes with nucleic acid encoding prostate carcinoma tumor antigen-1 wherein specifically hybridized probe produces a signal; and

d. detecting the presence of said signal as indicative of the presence of nucleic acid encoding prostate carcinoma tumor antigen-1, wherein the presence of said nucleic acid encoding prostate carcinoma tumor antigen-1 is indicative of the presence of metastatic prostate cancer cells in the human subject's blood.